



Methicillin-Resistant *Staphylococcus Aureus* in Hospitals and Communities: Awareness and Management of a Growing Concern

Learning Objectives

After completing this activity, participants should be better able to:

- Describe the prevalence and clinical significance of methicillin-resistant *Staphylococcus aureus* (MRSA) infections
- Determine the method for diagnosis of MRSA in hospital and community settings
- Define current practices in the treatment of key MRSA infections
- Recommend appropriate anti-MRSA regimens from available antibiotics

Introduction

Bacterial resistance to antimicrobial drugs has been a clinical and public health concern since the dawn of the golden age of antimicrobial discovery. Within 2 years of the introduction of penicillin in the early 1940s, penicillin-resistant strains of nosocomial staphylococci had been reported, and by the 1950s, penicillinase-producing strains of *Staphylococcus aureus* were considered “universally present” in hospitals.¹ Isolates from the community were still mostly penicillin-susceptible at this point, but when the prevalence of resistant *S aureus* in hospitals soared after World War II, rates in the community soon followed. By the late 1960s, both hospital and community resistance rates hovered at 80% or above, and today most strains of *S aureus* are penicillin-resistant.^{1,2}

The era of methicillin resistance began like the penicillin story, but its timetable and current patterns of dissemination are its own. Methicillin was introduced in 1959 as the first semisynthetic penicillin with resistance to penicillinase; today this class of beta-lactams includes oxacillin, amoxicillin, flucloxacillin, and others. Reports of methicillin-resistant *S aureus* acquired in hospitals (healthcare-associated MRSA [HA-MRSA]) appeared just 2 years after the drug’s debut (Figure 1). Although the problem spread steadily, for many decades MRSA was confined mainly to urban medical centers. In the late 1990s, however, the prevalence of community-acquired (CA) MRSA began a steep ascent.^{1,3}

Novel approaches to therapy and prevention will become more and more important, especially with the diminishing availability of new “wonder drugs.”

MRSA in Hospitals and Communities: Awareness and Management of a Growing Concern

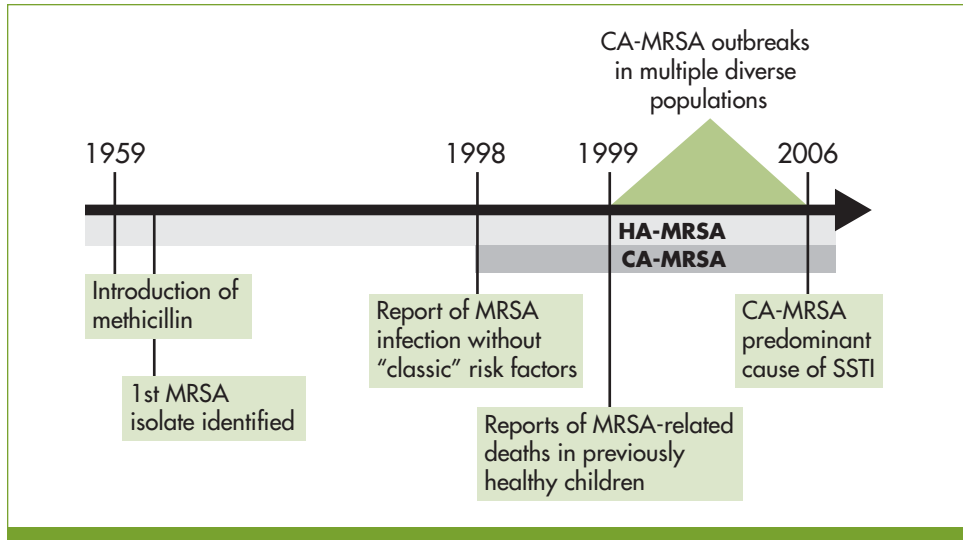


Figure 1. Trends in the prevalence of HA-MRSA and CA-MRSA.³

Changing Profile of *S Aureus* and MRSA

Today, MRSA is a growing concern in healthcare settings and the community, not only for its increasing prevalence but for its adaptations in favor of virulence and the 2-way dissemination pattern it seems to be following—hospital to community, and community to hospital, with stops along the way for adaptations and cloning.^{4,5} The current profile of *S aureus* and MRSA in America's hospitals and communities is best described by findings, such as the following, from major databases and surveillance programs:

- *S aureus* infections afflict 290,000 inpatients per year in US hospitals, resulting in 12,000 deaths, 2.7 million excess hospital days, and \$9.5 billion in excess charges (Nationwide Inpatient Sample database, Agency for Healthcare Research and Quality).⁶
- MRSA accounted for 64% of *S aureus* strains isolated from intensive care units in 2003—up strikingly from 36% in 1992 (National Nosocomial Infections Surveillance, Centers for Disease Control and Prevention).^{7,8}
- MRSA-related hospitalizations in short-stay hospitals increased by 119% between 1991 and 2005; MRSA-related septicemia and pneumonia (typically nosocomial) increased, but not as dramatically as skin and soft-tissue infections (SSTIs), which are typically community-acquired (National Hospital Discharge Survey).⁵
- CA-MRSA represented 8% to 20% of all MRSA isolates from inpatient and outpatient facilities at sites in Baltimore, Atlanta, and Minnesota in 2001 and 2002 (Centers for Disease Control's [CDC] MRSA Active Bacterial Core Surveillance [ABCs] program).⁹
- CA-MRSA was the most common cause of SSTIs in patients presenting to emergency departments in 11 US cities in 2004. Although more than 25% of patients had risk factors for HA-MRSA, 99% of the SSTI isolates were characteristic of CA-MRSA (EMERGENCY ID Net Study Group).¹⁰

Getting a clear and contemporary reading on the profile of MRSA in various settings is a challenge, but 2 factors are certain: the traditional divisions between “community” and “hospital” MRSA are changing; and *S aureus* resistance to methicillin will continue to spread. As with penicillin before it and the antibiotics that have followed it, “the question is not whether resistance will occur, but how prevalent resistance will become.”¹ In the midst of this evolution, clinicians in hospitals, primary care offices, outpatient surgical settings, and community healthcare settings carry an increasing responsibility to recognize and appropriately manage patients with possible or confirmed MRSA. New and existing guidelines can be of help, but clinical experience and informed judgment are fundamental to the task.

Community-Acquired or Hospital-Acquired MRSA? Differences and Why They Matter

Hospital-acquired MRSA—usually referred to today as healthcare-associated MRSA—traditionally has been defined as MRSA acquired in and related to a healthcare setting. Due to evidence that CA-MRSA is making its way into healthcare settings, HA-MRSA now is sometimes subclassified as having either community- or hospital-onset (Table 1).¹¹ Patients with community-onset infection have MRSA risk factors on presentation: a recent history of hospitalization, surgery, dialysis, or residence in a long-term care facility; a prior MRSA infection; or the presence of an invasive medical device. A recent study documented cases of MRSA in bedbugs examined from patients residing in areas with high rates of poverty and homelessness.¹² Hospital-onset cases are defined primarily by confirmation of MRSA more than 48 hours after admission. Community MRSA infections are defined as being acquired in the community in the absence of MRSA risk factors.¹¹ They are more likely than HA-MRSA infections to occur in children and young adults, ethnic groups, including blacks and Native Americans; athletes; individuals living in crowded conditions or in close contact with MRSA, and those who have used antibiotics frequently (Table 2).^{13,14}

Table 1.

Epidemiologic Classification of MRSA¹¹

	Onset Designation	Definition
HA-MRSA	Community-onset	Cases with a history of hospitalization, surgery or long-term care residence in the 12 months preceding culture date; prior MRSA infection; presence of an invasive device at time of admission
	Hospital-onset	Cases with positive cultures isolated more than 48 hours after hospital admission. Can also have community-onset risk factor(s)
CA-MRSA	Not specified	Cases with no community-onset risk factors

MRSA in Hospitals and Communities: Awareness and Management of a Growing Concern

MRSA experts note that from a clinical management standpoint formal differentiation of CA-MRSA from HA-MRSA is not as important as knowledge of local patterns of pathogen resistance.¹⁵ Nevertheless, there are noteworthy differences between the 2 main MRSA categories in terms of associated infections and susceptibility to antimicrobial therapy.

In hospitals, MRSA is the leading resistant pathogen responsible for conditions including ventilator-associated pneumonia, bacteremia from indwelling vascular catheters, surgical wound infections, and indwelling Foley catheter-associated urinary tract infections.¹⁶ Although CA-MRSA can and increasingly does present with severe manifestations such as necrotizing pneumonia or fasciitis,¹⁷ the majority of infections it causes are SSTIs such as furuncles and carbuncles (the lesions often are misidentified as spider bites). These may be superficial, but some are deep and require surgical and medical intervention in the hospital. Complicated SSTIs may result in serious disease, including bacteremia, pneumonia, osteomyelitis, purpura fulminans, epidemic furunculosis, and toxic shock syndrome.^{15,17}

Formal differentiation of CA-MRSA from HA-MRSA is not as important as knowledge of local patterns of pathogen resistance.

Table 2.

Risk Factors for HA-MRSA and CA-MRSA^{13,14}

Healthcare-Associated
<ul style="list-style-type: none">➤ Older age (elderly)➤ Prolonged hospitalization➤ Admission to intensive care unit (ICU)➤ Hemodialysis➤ Indwelling lines and catheters➤ Invasive procedures➤ Comorbid medical conditions
Community-Associated
<ul style="list-style-type: none">➤ Age extremes (children <2 years, elderly)➤ Certain ethnicities (Native American, black)➤ Crowded living conditions➤ Contact sports➤ IV drug use➤ Men who have intercourse with men➤ Household contact with person colonized or infected➤ Frequent antibiotic use, past MRSA infection➤ Veterinary work

IV = intravenous.

Isolates obtained from patients with a clinical diagnosis of CA-MRSA show genetic and other biologic differences compared with HA-MRSA isolates.¹⁸ The implications are not entirely clear, but these traits may underlie the observed differences in antimicrobial sensitivity between the MRSA types. In general, community-associated strains have been susceptible to a wider variety of antimicrobial drug classes than HA-MRSA strains. Indeed, many CA-MRSA isolates have shown resistance to only beta-lactams (penicillins and cephalosporins) and macrolides, while HA-MRSA isolates are resistant to multiple classes of drugs.¹⁵

Are Susceptibility Profiles Changing?

An important question is whether these susceptibility profiles might be changing with the changing dissemination patterns of MRSA, particularly the movement of CA-MRSA to health-care settings. Findings on the question vary. For example, Hultén and coauthors¹⁹ reported that

community-onset HA-MRSA resistance to clindamycin (widely used in SSTIs and in serious CA-MRSA infections in children) increased more than 4-fold (from 3.5% to 19%) between 2001 and 2004 at Texas Children's Hospital. By contrast, the Canadian Nosocomial Infection Surveillance Program found that MRSA strains from patients hospitalized between 1995 and 2008 became less resistant to clindamycin, as well as to tetracycline and trimethoprim-sulfamethoxazole (TMP-SMX). The strains were also highly susceptible to vancomycin and newer agents such as linezolid and daptomycin. These positive changes were attributed to the recent emergence of CA-MRSA in Canada; community strains generally were more susceptible than HA-MRSA in the surveillance results.²⁰

Suspect MRSA in the differential diagnosis of all purulent lesions.

Although CA-MRSA may be more susceptible to a broader range of antibiotics, in some cases it has presented in a more virulent form of *S aureus* than HA-MRSA.²¹ CA-MRSA infections may be severe and destructive despite culture-guided therapy, making it all the more critical that clinicians be fully informed of MRSA characteristics and management.

Importance of an Efficient and Accurate Diagnosis

As relevant as a patient's clinical and epidemiologic characteristics may be to the recognition of MRSA, these factors alone are not reliable guides to diagnosis and management of MRSA. In a prospective study of patients hospitalized with community-onset HA-MRSA, Miller and coauthors found the presenting risk factors for MRSA were poor predictors of MRSA (vs susceptible *S aureus* infections).²² Even patients without any risk factors or exposures had a relatively high probability of MRSA. The authors advised that risk factors not be used in decisions about empiric therapy or measures to control the spread of MRSA.²²

With this understanding, a reasonable approach to clinically identifying MRSA is to consider it in the differential diagnosis of all conditions compatible with *S aureus* infection, but especially infections of the skin and soft tissue. Any inflammation of the skin and subcutaneous tissue, an itchy rash (folliculitis), furuncles, carbuncles, pustules, impetigo, or abscess could involve MRSA.^{13,24} Experts recommend specifically that MRSA be included in the differential diagnosis

Shortening the Time to MRSA Diagnosis
Tests to identify MRSA are getting much faster. In 2008, the FDA approved an automated polymerase chain reaction (PCR) test that detects *S aureus* and MRSA from SSTI swabs in less than 1 hour. Cultures for susceptibility testing or epidemiologic typing still should be performed.²³

of all purulent lesions—that is, lesions with a fluctuant or palpable fluid-filled cavity, a yellow or white center, a central point or “head,” draining pus, or pus that can be aspirated with a needle and syringe (Figure 2).^{15,24} Many times these lesions are described as “spider bites,” a term that should immediately raise suspicion of *S aureus*. The role of *S aureus* in cellulitis without drainage or abscess is not clear because pathogens are rarely cultured in this condition.²⁴

Pain or symptoms distant from a cutaneous lesion may suggest systemic spread of infection.¹³ MRSA should be kept in the differential diagnosis of this and more severe and uncommon manifestations of *S aureus*, including sepsis syndrome, osteomyelitis, septic arthritis, severe pneumonia (or pneumonia

MRSA

MRSA in Hospitals and Communities:

Awareness and Management of a Growing Concern

following a flu-like illness, which has been associated with necrotizing pneumonia),¹⁴ necrotizing fasciitis, and purpura fulminans.¹⁵ In addition, it should be determined if these conditions are of community- or healthcare-associated onset.

Beginning Treatment

Incision and drainage is the first step in the treatment of purulent cutaneous lesions and septic joints. Management strategies encourage clinicians to use this procedure routinely, and to collect drainage specimens for culture and susceptibility testing to guide patient management and track local trends in *S aureus* susceptibility and resistance.^{15,24,25} Cultures to define invasive infections are appropriate; these may include blood from patients with signs of systemic infection, lung secretions or pleural fluid from patients with pneumonia, or specimens from other normally sterile sites such as joint or bone.¹⁵



Figure 2. Typical “spider bite” lesion compatible with *S aureus* infection and probably MRSA. Reproduced from Centers for Disease Control and Prevention.³

Guidelines for Treatment

Clinical practice guidelines for the treatment of MRSA have been developed by expert panels convened by the Centers for Disease Control (CDC)^{15,24} and the Infectious Diseases Society of America (IDSA), as well as experienced practitioners, institutional providers, and public agencies. The IDSA guidelines, published in January 2011, are the first to be issued by the society and were endorsed by the Pediatric Infectious Diseases Society, the American College of Emergency Physicians, and the American Academy of Pediatrics. Catherine Liu, MD, lead author of the IDSA guidelines, referred to them as a “framework” for evaluating and treating MRSA infection, and a “living document” meant to evolve as new information and antibiotics appear. Following are some of the document’s key recommendations^{26,27}:

- MRSA management should include identification, elimination, or debridement of the primary and other sites of infection—including drainage of abscesses, removal of central venous catheters, and debridement of osteomyelitis
- For most simple abscesses or boils, incision and drainage (I&D) alone without antibiotic therapy is probably adequate
- To document clearance of MRSA bacteremia, clinicians should obtain follow-up blood cultures 2 to 4 days after initial positive cultures and as needed thereafter
- In vitro susceptibility should be confirmed and documented when the clinician is considering alternatives to vancomycin in treating complicated SSTIs or invasive conditions
- In the absence of drug allergy, a beta-lactam antibiotic is the drug of choice for methicillin-susceptible *S aureus*

In particular, MRSA treatment guidelines fill knowledge gaps in treating the types of infections presenting most often at the frontlines of healthcare delivery: SSTIs and pneumonia.

SSTIs

Nonpurulent Infection

Given the still uncertain role of *S aureus* in nonpurulent cellulitis, guidelines on this condition recommend empiric therapy to target beta-hemolytic *Streptococcus* species or other suspected pathogens. Additional coverage for MRSA is recommended in patients who do not respond to beta-lactam therapy, and it may be considered in cases of systemic toxicity. Individualization of therapy should be based on the patient's responses during close follow-up.^{24,26}

Purulent Infection

A core principle in the management of abscesses and other purulent SSTIs is the importance of circumspect use of antimicrobial drugs. Incision and drainage is the primary therapy for furuncles, other abscesses, and septic joints, and the IDSA guidelines note that with simple abscesses, this therapy is likely to be adequate. Beyond simple lesions, however, antimicrobial therapy should be considered, with MRSA clearly in mind as a target. Among purulent SSTIs in patients presenting to emergency rooms in the EMERGENCY ID Net study, 59% were MRSA infections.¹⁰

The decision to use antimicrobial therapy is generally the clinician's judgment call based on (1) severity and speed of progression of the infection and presence of cellulitis; (2) signs and symptoms of systemic illness; (3) comorbidities or immune suppression; (4) age: very young or old patient; (5) location makes the abscess difficult to drain completely (such as the hand or genitals); (6) associated septic phlebitis; and (7) lack of response to I&D alone (Table 3).²⁶ While case-specific cultures are pending, treatment choices should be informed by local prevalence and resistance data regarding *S aureus* and other pathogens. Possible outpatient options for empirical MRSA therapy include^{15,17,24,26}:

- Clindamycin—widely, and thus far effectively, used in *S aureus* SSTIs as well as invasive infections in children
- A tetracycline such as doxycycline or minocycline—some data show effectiveness in SSTIs, but not against group A streptococcus. Minocycline is a possible alternative if resistance to doxycycline occurs. Not recommended for children younger than 8 years
- TMP-SMX—although most CA-MRSA strains are susceptible to TMP-SMX in vitro, data on clinical effectiveness is limited; like tetracyclines, there is no evidence of clinical effectiveness against group A strep. Not recommended for children younger than 8 weeks
- Linezolid—FDA-approved for MRSA SSTIs and pneumonia in adults and children. Its main adverse event is bone marrow suppression. Consider reserving this drug for more severe infections in consultation with infectious disease specialist.

Fluoroquinolones (ciprofloxacin, levofloxacin) and macrolides (erythromycin, clarithromycin, azithromycin) are not recommended treatment for MRSA SSTIs because resistance

Table 3.

MRSA: Indications for Antimicrobial Therapy After Incision and Drainage²⁶

- Severe or extensive disease (eg, involving multiple sites of infection) or rapid progression in presence of associated cellulitis
- Signs and symptoms of systemic illness
- Associated comorbidities or immunosuppression (diabetes mellitus, human immunodeficiency virus infection/AIDS, neoplasm)
- Extremes of age
- Abscess in area difficult to drain completely (eg, face, hand, genitalia)
- Associated septic phlebitis
- Lack of response to I&D alone

MRSA in Hospitals and Communities:

Awareness and Management of a Growing Concern

to these agents can develop rapidly and lead to relapse and treatment failure.^{15,24} Options for treatment of beta-hemolytic streptococci and CA-MRSA include clindamycin alone; TMP-SMX or a tetracycline in combination with a beta-lactam; or linezolid alone.²⁶

Complicated SSTIs

For hospital inpatients with a complicated SSTI, such as deeper soft-tissue infection, wound infection, major abscess, or an infected burn, recommended treatment involves surgical debridement, broad spectrum antibiotics, and empirical coverage of MRSA. Options for MRSA coverage include the following (Table 4)^{15,26}:

- Intravenous (IV) vancomycin
- Oral or IV linezolid
- IV daptomycin
- IV telavancin
- Oral or IV clindamycin

Treatment is recommended for 7 to 14 days for complicated SSTIs (as established in clinical trial protocols); however, treatment duration should be individualized on the basis of the patient's clinical response. Consultation with an infectious disease expert is advisable. None of the antibiotic options listed has shown consistent superiority to vancomycin in curing complicated SSTIs, although telavancin²⁸ and linezolid²⁹ have performed as effectively as vancomycin in clinical trials, and linezolid has been more effective with MRSA SSTIs specifically.²⁹ Use of tigecycline is not recommended in current guidelines; in 2010 the US Food and Drug Administration (FDA) issued a warning for this glycylicline drug based on an associated increase in mortality risk.³⁰

Other Measures

All patients with SSTIs should be educated about personal hygiene and wound protection (Table 5).¹⁵ Patients (or caretakers) should be instructed to keep draining wounds covered with clean, dry bandages; maintain good personal hygiene with regular bathing and hand-washing with soap and water or an alcohol-based gel; and avoid reusing or sharing personal items that have come in contact with infected skin.^{15,27}

Adults With Pneumonia

Both *S aureus* and MRSA are common pathogens in healthcare-associated pneumonia, including necrotizing pneumonia. *S aureus* in

Table 4.

Options for MRSA Coverage Pending Culture Data for Hospitalized Adult Patients With Complicated SSTI²⁶

Drug	Dose	Duration ^a
Vancomycin	15-20 mg/kg/dose IV every 8-12 h, not to exceed 2 g per dose	7-14 days
Linezolid	600 mg twice daily, oral or IV	7-14 days
Daptomycin	4 mg/kg IV once daily	7-14 days
Telavancin	10 mg/kg IV once daily	7-14 days
Clindamycin	600 mg IV or 300 mg orally 3 times daily	7-14 days

^aIndividualized per patient response during close follow-up.

Table 5.**Preventing SSTIs: Advice for Patients and Their Close Contacts**

1. Keep draining wounds covered with clean, dry, bandages
2. Clean hands regularly with soap and water or alcohol-based hand gel (if hands are not visibly soiled). Always clean hands immediately after touching infected skin or any item that has come in direct contact with the infected skin
3. Maintain good general hygiene with regular bathing
4. Do not share items that may be contaminated with wound drainage (eg, towels, clothing, bedding, razors, athletic equipment that touches the skin)
5. Promptly launder clothing that has come in contact with wound drainage; dry thoroughly
6. If the wound cannot be covered, avoid activities involving skin-to-skin contact with other persons (such as athletic activities) until the wound is healed
7. Clean equipment and other environmental surfaces with detergents and disinfectants specifically effective against *S aureus*

community-associated pneumonia is less common (about 3% of patients), but the majority of infections involve MRSA and are notable for their lethal virulence—a trait attributed to the gene toxin Panton-Valentine leukocidin, or PVL.^{31,32} They typically are preceded by flu-like illness in otherwise healthy children and adults. Necrotizing pneumonia may involve high fever, hemoptysis, hypotension, leukopenia, alveolar infiltrates that progress to abscesses (not typical of HA-MRSA), and life-threatening alveolar hemorrhage.¹⁷

A Word About Rifampin

Rifampin is often used as an adjunctive antimicrobial agent in *S aureus* infections, although clinical trial evidence to support this practice in MRSA infections is limited. Resistance to rifampin appears so rapidly that its use as monotherapy has long been discouraged. Drug interactions are also a concern.^{15,24} The recent IDSA guidelines recommend that rifampin not be used alone or as an adjunct in the treatment of skin and soft-tissue infections. In other selected MRSA scenarios, such as osteomyelitis and infective endocarditis, it may be used in combination with another *S aureus*-active agent.²⁶

Although not on the IDSA list of options for MRSA coverage in pneumonia, the ATTAIN study recently compared telavancin with vancomycin in patients with hospital-associated pneumonia, chiefly from MRSA. The drugs achieved nearly identical rates of clinical cures, although patients with mixed gram-positive/gram-negative pneumonia had better cure rates with vancomycin.³⁶

MRSA in Hospitals and Communities: Awareness and Management of a Growing Concern

IDSA Guidelines

The IDSA guidelines recommend empiric MRSA coverage for patients with pneumonia who require intensive care or have necrotizing or cavitary infiltrates or empyema. While sputum and/or blood cultures are pending, the following antimicrobials are treatment options for HA-MRSA or CA-MRSA pneumonia. Treatment is recommended for 7 to 21 days, depending on the extent of infection²⁶:

- ▶ IV vancomycin—vancomycin is the preferred therapy for severe and life-threatening MRSA infections. Recent findings from a randomized trial suggest rifampin in addition to vancomycin in patients with HA-MRSA pneumonia may improve antimicrobial efficacy. The combination was associated with higher clinical cure rates and lower mortality than vancomycin alone. However, while these differences consistently favored combination therapy, they were not all statistically significant. Moreover, there were no differences between treatment groups on rates of microbiologic eradication rates.³³
- ▶ Oral or IV linezolid—linezolid is an alternative to vancomycin. A randomized trial comparing this drug with vancomycin in patients with nosocomial pneumonia from *S aureus*, MRSA, or streptococcus found equivalence between the drugs in terms of clinical cure rates.³⁴ However, in a later retrospective analysis of this and other randomized trial data, the subset of patients with MRSA pneumonia had significantly better cure rates and survival with linezolid therapy: cure rate was 59% with linezolid and 36% with vancomycin ($P = .03$), while survival rates were 80% and 64%, respectively ($P < .01$).³⁵
- ▶ Oral or IV clindamycin—clindamycin is appropriate for selected patients with necrotizing pneumonia. There are few data regarding its specific effectiveness in adult MRSA pneumonia, although it has been used successfully to treat children with CA-MRSA pneumonia.²⁶

CASE: Healthy Woman With a Wound Infection: Management With MRSA Awareness



Presentation

Yvonne is a 50-year-old woman who recently had a vein-stripping procedure for severe varicose veins. She had the procedure as an outpatient and was back at work as a store clerk after a few days. Now she has presented to the emergency room with a 3-day history of fever and a purulent tender area on her lower left leg near her vein incision site. She reports no chills but says she feels fatigued. Yvonne has no other significant medical history; she is healthy and has been hospitalized in the past only for childbirth. She has been relieving her pain and fever with ibuprofen, which is her only medication.

Physical Examination

- Temperature: 101.2° F
- Blood pressure: 120/85 mm Hg
- Heart rate: 96 bpm
- Skin examination reveals a 7-cm area of raised, erythematous skin on the lower left leg. It is warm to the touch, mildly tender, and fluctuant. The remainder of the skin exam is normal
- No adenopathy in any area
- Trace pedal edema of the lower left leg
- Normal heart, lungs, and abdomen

Laboratory Findings

- White blood cell count: 12,000/mL (mildly elevated), with 90% neutrophils
- Normal platelet count (183,000/mL) and hematocrit (45%)

Clinical Decision Point

What is the first step in managing this patient's infection?

- Start Yvonne on oral antibiotic therapy
- Draw blood samples for culture
- Send her home with wound care instructions
- Call the surgeon to perform incision and drainage of abscess

Comment

The best next step in managing Yvonne's skin infection is I&D. Drawing blood cultures is appropriate because the patient has systemic symptoms. Nevertheless, proper I&D is the key to curing an abscess. Primary care and emergency department clinicians can easily master the technique for I&D; clinical training videos are widely available.³⁷ In the current case, however, it is best to call the surgeon who performed Yvonne's vein stripping.

Incision and drainage is performed and drainage samples are sent for culturing.

Clinical Decision Point

What is the best next step in managing Yvonne's infection?

- Draw blood cultures and admit Yvonne for antibiotic therapy with vancomycin, daptomycin, linezolid, or telavancin
- Send her home with wound care instructions only (I&D is sufficient)
- Send her home with topical antibiotics
- Send her home with prescription for cephalexin
- Send her home with prescription for linezolid

Comment

Neither wound care alone nor topical antibiotics alone would be appropriate for a patient with systemic symptoms of infection. Antibiotic therapy may not always be necessary along with I&D if an abscess is simple and the patient has no systemic symptoms or signs of deeper infection. However, if the decision is made to treat empirically, antibiotics should be selected with keen awareness of the prevalence of MRSA generally and in skin and soft-tissue infections specifically, whether community or healthcare associated. When patients do not appear toxic, a reasonable decision is to send them home with oral linezolid or another MRSA-active agent. Cephalexin, a beta-lactam, would not be an appropriate choice for MRSA coverage, though it is active against streptococcal pathogens.

In Yvonne's case, the presence of systemic signs of infection makes it prudent to admit her to the hospital and obtain blood and wound cultures (some clinicians admit all febrile patients with skin and soft-tissue injuries). Any of the antibiotics mentioned previously—vancomycin, daptomycin, linezolid, or telavancin—would be an appropriate choice for inpatient empiric MRSA coverage until sensitivities are obtained. Each has an A1 level of recommendation in the recently issued IDSA treatment guidelines.²⁶

Yvonne's infection, cultured as MRSA, responded well to a course of IV vancomycin. She was discharged after a week with educational materials on MRSA and measures to prevent its spread in the family and community.

References

1. Chambers HF. The changing epidemiology of *Staphylococcus aureus*? *Emerg Infect Dis*. 2001;7:178-182.
2. Lowy FD. Antimicrobial resistance: the example of *Staphylococcus aureus*. *J Clin Invest*. 2003;111:1265-1273.
3. Deresinski SC, Liu C, Talan DA. Community associated MRSA. In: Challenging Clinical Cases in Adult Immunizations: Focus on MRSA. AVindico Medical Education CME activity on InfectiousDiseaseNews.com. April 1, 2009. <http://www.infectiousdiseaseneews.com/article.aspx?id=42131#intro>. Accessed July 7, 2011.
4. Boyce JM. Community-associated methicillin-resistant *Staphylococcus aureus* as a cause of health care-associated infection. *Clin Infect Dis*. 2008;46:795-798.
5. Klein E, Smith D, Laxminarayan R. Hospitalizations and deaths caused by methicillin resistant *Staphylococcus aureus*, United States, 1999-2005. *Emerg Infect Dis*. 2007;13:1840-1846.
6. Noskin GA, Rubin RJ, Schentag JJ, et al. The burden of *Staphylococcus aureus* infections on hospitals in the United States. An analysis of the 2000 and 2001 Nationwide Inpatient Sample database. *Arch Intern Med*. 2005;165:1756-1761.
7. NNIS System. National Nosocomial Infections Surveillance (NNIS) system report, data summary from January 1992 through June 2004, issued October 2004. *Am J Infect Control*. 2004;32:470-485.
8. Klevens RM, Edwards JR, Tenover FC, McDonald LC, Horan T, Gaynes R; and National Nosocomial Infections Surveillance System. Changes in the epidemiology of methicillin-resistant *Staphylococcus aureus* in intensive care units in US hospitals, 1992-2003. *Clin Infect Dis*. 2006;42:389-391.
9. Fridkin SK, Hageman JC, Morrison M, et al; for Active Bacterial Core Surveillance Program of the Emerging Infections Program Network. Methicillin-resistant *Staphylococcus aureus* disease in three communities. *N Engl J Med*. 2005;352:1436-1444.
10. Moran G, Krishnadasan A, Gorwitz R, et al; EMERGEncy ID Net Study Group. Methicillin-resistant *S. aureus* infections among patients in the emergency department. *N Engl J Med*. 2006;355:666-674.
11. Klevens RM, Morrison MA, Nadle J, et al; for Active Bacterial Core Surveillance (ABCs) MRSA Investigators. Invasive methicillin-resistant *Staphylococcus aureus* infections in the United States. *JAMA*. 2007;298:1763-1771.
12. Lowe CF, Romney MG. Bedbugs as vectors for drug-resistant bacteria [letter]. *Emerg Infect Dis*. 2011;Jun [Epub ahead of print]
13. Kurkowski C. CA-MRSA: the new sports pathogen. *Orthopaed Nurs*. 2007;26:310-314.
14. Boucher HW, Corey GR. Epidemiology of methicillin-resistant *Staphylococcus aureus*. *Clin Infect Dis*. 2008;46:S344-S349.
15. Gorwitz RJ, Jernigan DB, Powers JH, Jernigan JA; Participants in the CDC Convened Experts' Meeting on Management of MRSA in the Community. Strategies for clinical management of MRSA in the community: Summary of an experts' meeting convened by the Centers for Disease Control and Prevention. 2006. http://www.cdc.gov/ncidod/dhqp/ar_mrsa_ca.html. Accessed July 7, 2011.
16. Hidron AI, Edwards JR, Patel J, et al; for National Healthcare Safety Network Team and Participating National Healthcare Safety Network Facilities. Antimicrobial-resistant pathogens associated with health-care-associated infections: annual summary of data reported to the national healthcare safety network at the Centers for Disease Control and Prevention, 2006-2007. *Infect Control Hosp Epidemiol*. 2008;29:996-1011.
17. Zetola N, Francis JS, Nuermberger EL, Bishai WR. Community-acquired methicillin-resistant *Staphylococcus aureus*: an emerging threat. *Lancet Infect Dis*. 2005;5:275-286.
18. Naimi TS, LeDell CH, Como-Sabetti K, et al. Comparison of community and health care-associated methicillin-resistant *Staphylococcus aureus* infection. *JAMA*. 2003;290:2976-2984.
19. Hultén KG, Kaplan SL, Gonzalez BE, et al. Three-year surveillance of community onset health care-associated *Staphylococcus aureus* infections in children. *Pediatr Infect Dis J*. 2006;25:349-353.

MRSA in Hospitals and Communities:

Awareness and Management of a Growing Concern

20. Simor AE, Louie L, Watt C, et al; and Canadian Nosocomial Infection Surveillance Program. Antimicrobial susceptibilities of health care-associated and community-associated strains of methicillin-resistant *Staphylococcus aureus* from hospitalized patients in Canada, 1995 to 2008. *Antimicrob Agents Chemother*. 2010;54:2265-2268.
21. Martinez JM. MRSA skin infection in athletes. Medscape Reference: Drugs, Conditions & Procedures. 2009. <http://emedicine.medscape.com/article/108972-overview>. Accessed July 7, 2011.
22. Miller LG, Perdreau-Remington F, Bayer AS. Clinical and epidemiologic characteristics cannot distinguish community-associated methicillin-resistant *Staphylococcus aureus* infection from methicillin-susceptible *S. aureus* infection: a prospective investigation. *Clin Infect Dis*. 2007;44:471-482.
23. Peterson LR. Molecular tests for RTI *S. aureus* and MRSA. FDA and IDSA workshop. November 13, 2009. www.fda.gov/downloads/MedicalDevices/NewsEvents/.../UCM199855.pdf. Accessed July 11, 2011.
24. Centers for Disease Control and Prevention (CDC). Outpatient flowchart. Outpatient management of MRSA skin and soft-tissue infections. <http://www.cdc.gov/mrsa/treatment/outpatient-management.html>. Accessed July 7, 2011.
25. Bamberger D, Boyd S. Management of staphylococcus aureus infections. *Am Fam Physician*. 2005;72:2474-2481.
26. Liu C, Bayer A, Cosgrove SE, et al. Clinical practice guidelines by the Infectious Diseases Society of America for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children. *Clin Infect Dis*. 2011;52:e18-e55.
27. Brooks M. IDSA issues first guidelines for treatment of MRSA. Medscape News Today. January 5, 2011. Available at: <http://www.medscape.com/viewarticle/735276>. Accessed July 7, 2011.
28. Stryjewski ME, Graham DR, Wilson SE, et al; for Assessment of Telavancin in Complicated Skin and Skin-Structure Infections Study. Telavancin versus vancomycin for the treatment of complicated skin and skin structure infections caused by gram positive organisms. *Clin Infect Dis*. 2008;11:1683-1693.
29. Weigelt J, Itani K, Stevens D, et al; and Linezolid CSSTI Study Group. Linezolid versus vancomycin in treatment of complicated skin and soft tissue infections. *Antimicrob Agents Chemother*. 2005;49:2260-2266.
30. FDA. FDA Drug Safety Communication: increased risk of death with Tygacil (tigecycline) compared to other antibiotics used to treat similar infections. Sept. 1, 2010. <http://www.fda.gov/Drugs/DrugSafety/ucm224370.htm>. Accessed July 7, 2011.
31. Hageman JC, Uyeki TM, Francis JS, et al. Severe community-acquired pneumonia due to *Staphylococcus aureus*, 2003-04 influenza season. *Emerg Infect Dis*. 2006;12:894-899.
32. Francis JS, Doherty MC, Lopatin U, et al. Severe community-onset pneumonia in healthy adults caused by methicillin-resistant *Staphylococcus aureus* carrying the Panton-Valentine leukocidin genes. *Clin Infect Dis*. 2005;40:100-107.
33. Jung YJ, Koh Y, Hong SB, et al. Effect of vancomycin plus rifampicin in the treatment of nosocomial methicillin-resistant *Staphylococcus aureus* pneumonia. *Crit Care Med*. 2010;38:175-180.
34. Rubinstein E, Cammarata SK, Oliphant TH, Wunderink RG; Linezolid Nosocomial Pneumonia Study Group. Linezolid (PNU-100766) versus vancomycin in the treatment of hospitalized patients with nosocomial pneumonia: a randomized, double-blind, multicenter study. *Clin Infect Dis*. 2001;32:402-412.
35. Wunderink RG, Rello J, Cammarata SK, Croos-Dabrera RV, Kollef MH. Linezolid vs vancomycin. *Chest*. 2003;124:1789-1797.
36. Rubinstein E, Lalani T, Corey GR, et al; ATTAIn Study Group. Telavancin versus vancomycin for hospital-acquired pneumonia due to gram-positive pathogens. *Clin Infect Dis*. 2011;52:31-40.
37. Fitch MT, Manthey DE, McGinnis HD, Nicks BA, Pariyadath M. Videos in clinical medicine: Abscess incision and drainage. *N Engl J Med*. 2007;357:e20. November 8, 2007. <http://www.nejm.org/doi/full/10.1056/NEJMvcm071319>. Accessed July 7, 2011.

To view **Frequently Asked Questions About MRSA** go to www.practicingclinicians.com/2011hs2